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6	2	fluoresc\$4 same polari\$7 same steroid same receptor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/14 10:59
7	5	fluoresc\$4 same polari\$7 same estrogen same receptor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/14 10:59

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L2 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:190219 CAPLUS

TITLE: High throughput fluorescence polarization-based screening assays for the identification of novel nuclear receptor ligands

AUTHOR(S): Eliason, Hildegard C.; Shekhani, Mohammed Saleh; Ervin, Kerry M.; Halbleib, Cale M.; Millis, Sherri Z.;

Mei, Baigen; Lowery, Robert G.; Burke, Thomas J. PanVera Corp., Madison, WI, 53719, USA

SOURCE: Abstracts of Papers, 223rd ACS National Meeting, Orlando, FL, United States, April 7-11, 2002 (2002), MEDI-100. American Chemical Society: Washington, D. C.

CODEN: 69CKQP

DOCUMENT TYPE: Conference; Meeting Abstract

LANGUAGE: English

AB Steroid hormone receptors (SHRs) are ligand-induced transcription factors that mediate the transactivation of genes responsible for cellular differentiation, reprodn., and metab. PanVera has developed a panel of fluorescence polarization (FP)-based high throughput screening assays for the rapid identification of novel SHR ligands for androgen, progesterone, glucocorticoid, and estrogen (alpha and beta) receptors. These homogeneous assays utilize recombinant human receptor proteins and fluorophore-steroid conjugates specific for these receptors. The synthetic fluorescent ligands bind with affinities similar to that of their resp. native ligands - generally in the low nanomolar range.

In FP assays, the polarization of the fluorophore is proportional to the fraction complexed with receptor. One can deduce the binding affinity of a test compd. by measuring its ability to

displace a **fluorescent** ligand from the **receptor's** hormone binding pocket. Such screening assays provide a simple and rapid method for detecting novel SHR ligands for this important class of drug targets.

L2 ANSWER 2 OF 8 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000227100 MEDLINE
DOCUMENT NUMBER: 20227100 PubMed ID: 10766033
TITLE: Modulation of LH/hCG receptors and physical state of ovarian membranes in rat pseudopregnancy.
AUTHOR: Jezova M; Scsukova S; Vranova J; Kolena J
CORPORATE SOURCE: Institute of Experimental Endocrinology, Slovak Academy of Sciences, Bratislava.. ueenjez@savba.savba.sk
SOURCE: GENERAL PHYSIOLOGY AND BIOPHYSICS, (1999 Dec) 18 (4) 347-56.
Journal code: 8400604. ISSN: 0231-5882.
PUB. COUNTRY: Slovakia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000606
Last Updated on STN: 20000606
Entered Medline: 20000523

AB . . . as well as regression of corpora lutea. The effects of cyclooxygenase inhibitors (indomethacin and acetylsalicylic acid (ASA)) and of selected **steroids** (estradiol, testosterone and dihydrotestosterone) on the functional state of luteinized ovaries were studied. The compounds were administered to the animals. . . .
injection.
ASA and indomethacin administration on days 10 and 11 after hCG injection resulted in an increase in the LH/hCG **receptor** binding activity and rigidity of ovarian membrane lipids, as determined by **fluorescence polarization** of 1,6-diphenyl-1,3,5-hexatriene (DPH) probe. This effect was apparent within 7 days after indomethacin and ASA treatment. Both estradiol and. . . Unlike testosterone, the administration of dihydrotestosterone induced a decrease
in membrane lipid rigidity and reduced the accessibility of the LH/hCG **receptor**. Inhibitors of prostaglandin F2alpha (PGF2alpha) synthesis, as the endogenous mediator of luteolysis, were shown to delay the regression of the. . . .

L2 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:112498 CAPLUS
DOCUMENT NUMBER: 128:176476
TITLE: A method for quantitating competitive binding of molecules to **steroid** hormone **receptors** utilizing **fluorescence polarization**
INVENTOR(S): Bolger, Randall E.; Ervin, Kerry M.; Lowery, Robert G.; Checovich, William J.
PATENT ASSIGNEE(S): Panvera Corp., USA; Burke, Thomas, J.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9805962	A1	19980212	WO 1997-US13538	19970801
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
PRIORITY APPLN. INFO.:			US 1996-23034P	P 19960802

TI A method for quantitating competitive binding of molecules to
steroid hormone receptors utilizing fluorescence polarization

AB The system comprises mixing a fluorescence-emitting compd. that
binds to the steroid hormone receptors, particularly
the estrogen receptor, in a soln. contg. the steroid
hormone receptors. Then, measuring the fluorescence
polarization of the soln. Subsequently, incubating the soln. with
at least one mol. that may compete with the compd. for interaction with
the steroid hormone receptors. Measuring the
fluorescence polarization of the soln. again. Finally,
comparing the fluorescence polarization measurements
to quantify any competitive interaction. A fluorescence
-emitting compd. such as a fluorescence-emitting hormone can be
used in combination with a fluorophore covalently coupled to an
oligonucleotide to study how hormone and oligonucleotide binding to the
hormone receptor are affected by each other.

ST steroid receptor compd binding fluorescence
polarization

IT Nucleic acids
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(fluorescence-labeled; method for quantitating competitive
binding of mols., including nucleotides, to steroid hormone
receptors utilizing fluorescence polarization
)

IT DNA
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(labeled with fluorescein; method for quantitating
competitive binding of mols., including nucleotides, to steroid
hormone receptors utilizing fluorescence
polarization)

IT Polarized fluorescence
(method for quantitating competitive binding of mols. to
steroid hormone receptors utilizing
fluorescence polarization)

IT Estrogen receptors
Estrogens
Steroid receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(method for quantitating competitive binding of mols. to
steroid hormone receptors utilizing
fluorescence polarization)

IT DNA
Nucleic acids
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(method for quantitating competitive binding of mols., including
nucleotides, to steroid hormone receptors utilizing
fluorescence polarization)

IT Estrogen receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(recombinant; method for quantitating competitive binding of mols.,
including nucleotides, to steroid hormone receptors
utilizing fluorescence polarization)

IT 18930-97-7D, 5,6,11,12-Tetrahydrochrysene, derivs.
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(fluorescence emitting hormone; method for quantitating
competitive binding of mols. to steroid hormone
receptors utilizing fluorescence polarization
)

IT 50-28-2, Estradiol, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(method for quantitating competitive binding of mols. to

steroid hormone receptors utilizing
fluorescence polarization)
IT 2321-07-5D, Fluorescein, DNA labeled with
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(method for quantitating competitive binding of mols., including
nucleotides, to steroid hormone receptors utilizing
fluorescence polarization)

L2 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1997:395830 CAPLUS

DOCUMENT NUMBER: 127:107177

TITLE: Phospholipase C inhibitor, U73122, releases
intracellular Ca²⁺, potentiates Ins(1,4,5)P₃-mediated
Ca²⁺ release and directly activates ion channels in
mouse pancreatic acinar cells

AUTHOR(S): Mogami, Hideo; Mills, Chris Lloyd; Gallacher, David
V.

CORPORATE SOURCE: The Physiological Lab., Liverpool, L69 3BX, UK

SOURCE: Biochemical Journal (1997), 324(2), 645-651

CODEN: BIJOAK; ISSN: 0264-6021

PUBLISHER: Portland Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It is recognized in many cellular systems that the **receptor**
/G-protein activation of phospholipase C and Ins(1,4,5)P₃ prodn. is the
transduction pathway regulating the release of Ca²⁺ from internal stores.
Ca²⁺ signals can now be monitored at the level of single cells but the
biochem. detection of Ins(1,4,5)P₃ cannot match this resoln. It is often
difficult or impossible to directly attribute responses evoked in single
cells by putative phospholipase C-coupled agonists to changes in
Ins(1,4,5)P₃ levels. U 73122 is an amino **steroid** that is
reported to act as a specific inhibitor of phospholipase C and it has
become an important tool in establishing the link between phospholipase C
activation and cellular Ca²⁺ signaling. In the present study we use both
patch-clamp electrophysiol. and the imaging of **fluorescent** Ca²⁺
indicators to investigate the effect of U 73122 in mouse pancreatic

acinar

cells. The study reveals that U 73122 has effects other than the
inhibition of phospholipase C. U 73122 can directly activate ion
channels. It can itself promote the release of Ca²⁺ from intracellular
stores in permeabilized cells and in intact cells it triggers a release

of

Ca²⁺ that is initiated specifically at the secretory pole of these
morphol. and functionally **polarized** cells. We also present
evidence that U 73122 can potentiate the response to Ins(1,4,5)P₃; this

is

seen both in permeabilized cells and in patch-clamp protocols in which
cells are internally dialyzed with submaximal concns. of Ins(1,4,5)P₃.
The effects of U 73122 are therefore multiple and not specific for the
inhibition of phospholipase C. Importantly, all the effects described
influence Ca²⁺ signaling yet in many exptl. protocols some of these
effects can go unnoticed and might in error be attributed simply to the
inhibition of Ins(1,4,5)P₃ prodn.

L2 ANSWER 5 OF 8

MEDLINE

DUPLICATE 2

ACCESSION NUMBER: 86221226 MEDLINE

DOCUMENT NUMBER: 86221226 PubMed ID: 3011559

TITLE: Sex steroid and prostaglandin interactions upon the
purified rat myometrial plasma membranes.

AUTHOR: Delicostantinos G; Fotiou S

SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1986 May) 45 (2-3)
149-56.

Journal code: 7500844. ISSN: 0303-7207.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198607
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19860710

AB . . . concentration of 1×10^{-6} M for 1 h at 37 degrees C, bind into MPM at pmolar concentrations. Unlabeled **steroids** inhibited [3H]PGE2 and [3H]PGF2 alpha binding to MPM in a dose-dependent manner. Membrane-bound and free **steroids** or PGs were found to be essentially unchanged under the present incubation conditions. Ca^{2+} ions up to 10 mM increased **steroid** binding into MPM. Molecular interactions between **steroids** and MPM were assessed by measuring the steady-state **fluorescence polarization** of 1,6-diphenyl-1,3,5-hexatriene (DPH), and by estimating the changes in the allosteric properties of MPM-bound ($\text{Na}^+ + \text{K}^+$)ATPase by fluoride (F^-). **Steroids** appear to increase the MPM fluidity, evaluated through changes in the Hill coefficient for MPM-bound ($\text{Na}^+ + \text{K}^+$)ATPase by F^- and by the **fluorescence polarization** method. Binding of sex **steroids** to MPM increased the membrane fluidity and decreased the binding of the uterus stimulatory PGs by membrane **receptors**. These studies provide a basis for postulating that a 'non-genomic' mechanism of sex **steroids** induces reduction of uterine contractions.

L2 ANSWER 6 OF 8 MEDLINE

ACCESSION NUMBER: 77159867 MEDLINE
DOCUMENT NUMBER: 77159867 PubMed ID: 856460
TITLE:

Fluidity of membrane lipids and lateral mobility of concanavalin A receptors in the cell surface of normal lymphocytes and lymphocytes from patients with malignant lymphomas and leukemias.

AUTHOR: Ben-Bassat H; Polliak A; Rosenbaum S M; Naparstek E; Shouval D; Inbar M

SOURCE: CANCER RESEARCH, (1977 May) 37 (5) 1307-12.
Journal code: 2984705R. ISSN: 0008-5472.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 197706

ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770622

AB . . . with nonmalignant and malignant disorders were studied for fluidity of membrane lipids and lateral mobility of concanavalin A (Con

A)

receptors. The degree of fluidity of the surface membrane lipid core was monitored quantitatively by **fluorescence polarization** analysis using the probe 1,6-diphenyl-1,3,5-hexatriene embedded in lipid regions of the surface membrane of intact cells. Mobility of Con A surface **receptors** was determined by the cap-forming ability after binding of **fluorescent** Con A. The present studies were performed on lymphocytes from 28 patients with malignant lymphomas, 22 patients with leukemia, 28. . . fluidity was less pronounced in lymphocytes isolated from leukemic patients in

clinical

remission and from leukemic patients receiving treatment with **steroids**. The results also show a marked difference in the cap-forming ability of lymphocytes from patients with malignant lymphomas or leukemia. . . a higher cap-forming ability. The cap-forming ability of cells from patients with chronic lymphocytic leukemia was unaffected

by

treatment with **steroids**. The present results, which are in line with previous observations, have shown that normal lymphocytes can be characterized by a low degree of lipid fluidity but a high degree of mobility of Con A **receptors**, whereas leukemic lymphocytes are

characterized by a high degree of lipid fluidity but a low degree of mobility of **Con A receptors**. These results confirmed our general hypothesis on the dynamic interrelation between membrane lipids and membrane protein **receptors**, and they indicate that the widely accepted term "membrane fluidity" requires better consideration for different membrane components.

L2 ANSWER 7 OF 8 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
 ACCESSION NUMBER: 78105430 EMBASE
 DOCUMENT NUMBER: 1978105430
 TITLE: Fluidity of membrane lipids and lateral mobility of concanavalin A receptors in the cell surface of normal lymphocytes and lymphocytes from patients with malignant lymphomas and leukemias.
 AUTHOR: Ben Bassat H.; Polliak A.; Rosenbaum S.M.; et al.
 CORPORATE SOURCE: Dept. Hematol. Med. A, Chanock Cent. Virol., Hebrew Univ. Hadassah Med. Sch., Jerusalem, Israel
 SOURCE: Cancer Research, (1977) 37/5 (1307-1312).
 CODEN: CNREA8
 COUNTRY: United States
 DOCUMENT TYPE: Journal
 FILE SEGMENT: 016 Cancer
 025 Hematology
 005 General Pathology and Pathological Anatomy
 026 Immunology, Serology and Transplantation

LANGUAGE: English

AB . . . with nonmalignant and malignant disorders were studied for fluidity of membrane lipids and lateral mobility of concanavalin A (Con A)

receptors. The degree of fluidity of the surface membrane lipid core was monitored quantitatively by **fluorescence polarization** analysis using the probe 1,6-diphenyl-1,3,5-hexatriene embedded in lipid regions of the surface membrane of intact cells. Mobility of Con A surface **receptors** was determined by the cap-forming ability after binding of **fluorescent** Con A. The present studies were performed on lymphocytes from 28 patients with malignant lymphomas, 22 patients with leukemia, 28. . . fluidity was less pronounced in lymphocytes isolated from leukemic patients in

clinical

remission and from leukemic patients receiving treatment with **steroids**. The results also show a marked difference in the cap-forming ability of lymphocytes from patients with malignant lymphomas or leukemia. . . a higher cap-forming ability. The cap-forming ability of cells from patients with chronic lymphocytic leukemia was unaffected

by

treatment with **steroids**. The present results, which are in line with previous observations, have shown that normal lymphocytes can be characterized by a low degree of lipid fluidity but a high degree of mobility of Con A **receptors**, whereas leukemic lymphocytes are characterized by a high degree of lipid fluidity but a low degree of mobility of Con A **receptors**. These results confirmed the general hypothesis on the dynamic interrelation between membrane lipids and membrane protein **receptors**, and they indicate that the widely accepted term 'membrane fluidity' required better consideration for different membrane components.

L2 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 ACCESSION NUMBER: 1977:205439 BIOSIS
 DOCUMENT NUMBER: BA64:27803
 TITLE: FLUIDITY OF MEMBRANE LIPIDS AND LATERAL MOBILITY OF CONCAVALIN A RECEPTORS IN THE CELL SURFACE OF NORMAL LYMPHOCYTES AND LYMPHOCYTES FROM PATIENTS WITH MALIGNANT LYMPHOMAS AND LEUKEMIAS.
 AUTHOR(S): BEN-BASSAT H; POLLIACK A; ROSENBAUM S M; NAPARSTEK E; SHOUVAL D; INBAR M
 SOURCE: CANCER RES, (1977) 37 (5), 1207-1312.

FILE SEGMENT:

BA; OLD

LANGUAGE:

Unavailable

AB. . . with nonmalignant and malignant disorders were studied for fluidity of membrane lipids and lateral mobility of concanavalin A (Con A) **receptors**. The degree of fluidity of the surface membrane lipid core was monitored quantitatively by **fluorescence polarization** analysis using the probe 1,6-diphenyl-1,3,5-hexatriene embedded in lipid regions of the surface membrane of intact cells. Mobility of Con A surface **receptors** was determined by the cap-forming ability after binding of **fluorescent** Con A. The present studies were performed on lymphocytes from 28 patients with malignant lymphomas, 22 patients with leukemia, 28. . . membrane fluidity was less pronounced in lymphocytes isolated from leukemic patients in clinical remission and leukemic patients receiving treatment with **steroids**. The results also show a marked difference in the cap-forming ability of lymphocytes from patients with malignant lymphomas or leukemia. . . a higher cap-forming ability. The cap-forming ability of cells from patients with chronic lymphocytic leukemia was unaffected

by

treatment with **steroids**. The present results, which are in line with previous observations, showed that normal lymphocytes can be characterized by a low degree of lipid fluidity but a high degree of mobility of Con A **receptors**, whereas leukemic lymphocytes are characterized by a high degree of lipid fluidity but a low degree of mobility of Con A **receptors**. These results confirmed the general hypothesis on the dynamic interrelation between membrane lipids and membrane protein **receptors**, and they indicate that the widely accepted term membrane fluidity requires better consideration for different membrane components.

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